EXHIBIT 13

EXPERT REPORT OF VICTOR L. ROGGLI, M.D.

Report for David T. Austern,
Future Claimants' Representative

In re W.R. Grace & Co., *et al.*

June 11, 2007

Dr. Epstein further states that asbestosis rarely occurs in individuals without plaques. I have found plaques in over 80% of my cases of histologically proven asbestosis. However, plaques found at autopsy are frequently missed premortem (i.e., radiographically). I have found that plaques found at autopsy were observed on plain films by an experienced thoracic radiologist in only 28% of cases.[3]

Dr. Epstein and Dr. Weill each comment upon the relationship between asbestosis and lung cancer, one concluding that asbestosis is an obligate precursor and the other that it is not. This issue has been reviewed in great detail by Henderson, et al.[4] These authors looked at all aspects of asbestos carcinogenesis (epidemiology, pathology, mineral fiber analyses, experimental animal studies, and in vitro studies) and reviewed nearly 400 references on the topic. They concluded that asbestosis is not an obligate precursor of asbestos-induced lung cancer and that the total dose of asbestos is the determining factor for an asbestos contribution to causation. These findings have since been reinforced by me and others.[5,6]

Dr. Weill and Dr. Moolgavkar opine that the rates of mesothelioma among women have been constant and that these represent a background rate of the disease. The implication is that mesotheliomas occurring in women are not asbestos-related. The fallacy of this is clear when you consider that I have observed a woman with mesothelioma whose only known exposure was washing the clothes of her insulator husband, and that this woman had (in addition to mesothelioma) histologically proven pleural plaques, asbestosis, and a lung fiber burden 10,000 times higher than that of an unexposed population. Indeed, my studies have found that approximately 70% of mesotheliomas in women are asbestos-related, and that over half are related to exposures as household contacts of asbestos exposed workers.[7,8]

Dr. Garabrant launches a vitriolic attack on the Helsinki criteria, published in 1997 in the Scandinavian Journal of Work, Environment and Health.[9] He notes that the document has no citations to the literature. Apparently, he is unaware that the publication he criticizes is an abridged version of a document called Asbestos, Asbestosis and Cancer published by the Finnish Institute of Occupational Health and supported by hundreds of references to peer-reviewed publications.[10] With respect to lung cancer, Dr. Garabrant states that no threshold value for lung asbestos content that permits a conclusion about lung cancer causation has been defined. This is incorrect.[6,11] The claim that any mesothelioma case in which an asbestos exposure can be recalled can be related to asbestos by the Helsinki criteria misstates the intention of the document. My own studies have shown that 94% of mesotheliomas in the United States have exposures to asbestos in one of 12 industrial settings (with one exception), one of 6 occupations, or one non-occupational setting.[8] The twelve industries include shipbuilding, US Navy/merchant marine, construction, insulation, power plants, railroad, oil/chemical, molten metal, asbestos manufacturing, paper mills, and ceramics/glass. The one exception is the automotive industry, for which there is abundant evidence that asbestos exposure in this industry does not cause asbestos-related disease.[12] The six occupations include pipefitters/welders, boiler workers, electricians, sheet metal workers, maintenance workers, and machinists. The one non-occupational setting is household contacts of asbestos workers. Finally, Dr. Garabrant states that extrapolation of exposures to low levels must be done with extreme caution. Although it is quite likely that there are

thresholds of exposure for the development of asbestosis and lung cancer, it should be noted that no level of asbestos exposure has yet been identified below which mesothelioma will not occur in humans.[13]

Dr. Moolgavkar states that there is no evidence that Libby fibers are any more toxic than other amphiboles. This statement offers little comfort considering the track record of crocidolite and amosite asbestos,[6-8, 11-14] and is analogous to the statement that the atomic bomb is no more dangerous than the hydrogen bomb. He also states that there are other causes of mesothelioma besides asbestos, and goes on to list radiation, erionite fibers, and SV40 as probable or possible causative agents. Radiation in my experience accounts for less than 1% of cases, erionite has never been identified as a causative agent of a mesothelioma case from the United States, and SV40 has largely been discredited as a cause of mesothelioma in humans.[11] Finally, he refers to the Peto model as indicating that earlier exposures have a greater attributable risk to mesothelioma than later exposures in an individual case. There are strong biological reasons that the Peto model should not be applied to multiple exposures for an individual case of mesothelioma. Whereas each individual has a unique latency period (i.e., the interval from first asbestos exposure to the diagnosis of disease), the application of the Peto model to multiple exposures for an individual involves assumption of multiple latency periods, which makes no sense for an individual (you *can* have multiple latency intervals in a *population* of exposed workers). Indeed, the mathematical formulas used in this way require the assumption that each separate exposure must interact with mesothelial cells that are at the very beginning of the process of conversion to neoplastic cells, and this is by no means the case (a fiber could just as well interact with a mesothelial cell which has already undergone some alterations in the process of carcinogenesis).

Dr. Rodricks offers a state-of-the-art review of knowledge about asbestos and disease, particularly as it relates to threshold limit values (TLVs) and permissible exposure limits (PELs), which he sometimes describes as 'safe' or 'protective.' It should be recognized that TLVs and PELs have always been compromise workplace levels, and were designed to try to minimize, but not absolutely prevent, the development of asbestosis.[15, 16] They were never expressly designed to prevent malignancy. In this regard, it should be noted that exposure to crocidolite asbestos at the current PEL of 0.1 fiber/cc for two months will double the risk of mesothelioma, assuming a linear dose response no threshold model.[17]

Dr. Richard J. Lee also discusses fiber release from surfaces (see discussion of Dr. Bragg's report above), and states that routine maintenance of asbestos-containing materials does not result in elevated exposures. I have reported a case of malignant mesothelioma in a teacher's aide whose only known exposure was to asbestos-containing ceiling tiles in the school in which she worked. Lung fiber analyses confirmed an increased level of tremolite asbestos.[18] The ceiling tile contained a considerable percentage of Canadian chrysotile asbestos, which is known to be contaminated with tremolite. I have also found that individuals with mesothelioma whose primary occupation was as a maintenance worker or maintenance mechanic had average asbestos body counts 20 times higher than our upper limit of normal.[8]